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Passive Smoking in Females and Coronary Heart Disease

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ABSTRACT

Thirty four cases of women with coronary heart disease (CHD) (22 cases diagnosed by coronary arteriography and 12 cases diagnosed as having myocardial infarction) were used in an investigation designed to assess the association between passive smoking in women and the establishment of CHD. The odds ratio (OR) of non-smoking women developing CHD as a result of exposure to passive smoke is 3.0-3.5 ($p<0.05$). A dose response relationship was detected between the number of passive smoke exposure years and the increase in OR for CHD. Multiple regression analysis shows that of the many risk factors for CHD, passive smoke exposure is significantly correlated with CHD. Women exposed to passive smoke also showed abnormal levels of serum LDL-C, HDL-C, apoA1 and apoB.

Key Words

Coronary heart disease, coronary arteriography, passive smoke

Experimental investigations have demonstrated that the chemical constituents generated in the sidestream smoke often contain the same harmful chemicals as in mainstream smoke inhaled by smokers, and that there is considerable adverse effects contributed by sidestream smoke to non-smokers who are passively exposed.[1,2] A number of reports have appeared showing a correlation between passive smoke and the damage to lung functions, increased incidence of lung cancer, and angina pectoris.[3-6] A limited number of investigations have been focused on the subject of passive smoke in the People's Republic of China, and have only concentrated on studying the influence of passive smoking on lung functions.[7] In this communication we report the relationship between passive smoke exposure and female patients who were hospitalized because of coronary heart disease.

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Materials and Methods

Subjects consisted of patients hospitalized between 1985-1987 and diagnosed as having coronary heart disease and myocardial infarction. They were matched employing the 1:2 method as follows: those who were admitted because of possible coronary heart disease and were later confirmed by coronary arteriography to be normals; patients with endocrine dysfunctions but free of CHDs; and people randomly selected from the population. Thus three groups were included in the present investigation: Group 1, those hospitalized and diagnosed with CHDs or myocardial infarction; Group 2, those hospitalized with endocrine problems but having no symptoms of CHDs, and Group 3, normals from the general population.

Each of the subjects in the three groups was interviewed using a standardized questionnaire. Some of the questions addressed included : subjects and the spouses smoking history, the age at which smoking began, the average daily cigarette consumption. Active and passive smoking were defined as follows : 1. Smoking at least one cigarette per day for a period of at least one year. The spouse is defined as an ex-smoker if he has already stopped smoking at least 5 years at the time of interview. 2. Wife who is a non-smoker but has lived with a smoking husband for at least 5 years is classified as a passive smoker. 3. If husband is a smoker before marriage, the wife exposure begins at time of marriage. Alternatively, the wife can become exposed after marriage if the husband picks up the smoking habit after marriage. Total exposure time is determined by divorce, death of husband, or when the husband quits smoking and becomes an ex-smoker. 4. Single female is considered to be equivalent to a female without a smoking spouse. To verify the accuracy of the data collected by the structured interview, tape recording was used and randomized re-interview was performed.

Subject group consist of 34 cases (22 cases diagnosed with CHD, and 12 cases diagnosed with myocardial infarction). Control group consist of 34 hospitalized subjects (with 13 suspected of CHDs but later confirmed to be normals) and 34 randomly selected matched for race, occupation, residence and age (+/- 5 years). Multiple regressional analysis was performed and the data analyzed suing a Sun-68000 electronic calculator.[9]

Results

1. Comparison between the Subject and the Control Groups

No significant differences exist between the two groups in regard to age, education, the marriage age. The mean ages of the diseased and control groups are 53.7±4.28 and 52.93±5.24, respectively (t=1.282, p>0.05)

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2. Effects of Passive Smoking

Table 1 shows a comparison between disease and paired control groups. The OR of getting CHD for nonsmoking women living with a smoking husband is 3.00, with a 95% CI of 1.256-7.168, i.e. the risk of women getting CHD is 3 times higher for those with husbands that smoke compared to those with nonsmokers husbands.

Table 1. A comparison of Passive Smoking Status in Diseased and Control Groups

		Control Group		
		+ +	+ -	- -
Diseased Group	+	4	12	9
	-	3	4	2
OR		3.00		
OR 95% CI		1.256-7.168		
X^2 (P)		6.117	(< 0.05)	

A. Dose Response Relationship

Table 2 illustrates the association between husbands' average daily cigarette consumption, passive smoke exposure years, cumulative passive smoke amount index, and the ORs of getting CHD. There is a noticeable dose response relationship, i.e. as the amount of passive smoke exposure increases, the risk of getting CHD also becomes greater.

Table 2. Dose Response Relationship between Passive Smoke Exposure and CHDs

	Subject	Control	OR	X^2
Husbands daily cigarette consumption				
0	9	38	1.000	.....

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<20	12	22	2.303	1.880
>20	13	8	6.861	10.098**
Passive Smoke Exposure Years				
0	9	38	1.000	.....
≤10	4	9	1.877	0.266
≤20	8	11	3.071	2.581
>20	13	10	5.489	8.230**
Cumulative Passive Smoke index (Years)				
0	9	38	1.000	.....
1-199	4	11	1.535	0.066
200-399	6	11	2.303	1.009
400-599	6	5	5.067	4.054*
600*	9	3	12.667	11.358***

*P<0.05 **P<0.01

B. Association with Clinical Diagnosis

In the patients group, 21 cases were diagnosed with angina pectoris and 13 cases with myocardial infarction. The number of passive smokers in both clinical settings is similar (X^2 = 1.298, p>0.5). These results are illustrated in Table 3. The results show that angina pectoris is clearly and significantly correlated with passive smoking. Although myocardial infarction in the passive smoking group show an OR of greater than 1, it did not reach statistical significance, which may be related to the small sample size.

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Table 3. Clinical Diagnosis in Passive Smoke Group

	Exposure to Passive Smoke		Exposure to Passive Smoke	
	Yes	No	Yes	No
	Angina Pectoris		Myocardial Infarction	
Subject Group	17	4	8	5
Control Group	20	22	10	16
OR	4.675		2.550	
X^2	5.035		1.018	
P	<0.05		>0.05	

C. Blood cholesterol and Lipoprotein Level Changes in Passive Smokers

By controlling for age, weight, and other risk factors, a decrease in serum HDL-C and apoA1 levels was found in passive smokers, whereas LDL-C, apoB and apo B/A1 levels are higher than those not exposed to passive smoke. The level of HDL-C, apoA1 and apoB/A1 levels are significantly different between the subject and control groups (Table 4)

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Table 4. Blood Cholesterol and Serum Lipoprotein Levels in Female Passive Smokers

	Control Group		CHD Group	
	Non-exposed	Exposed	Non-exposed	Exposed
Number	26	20	9	24
Total cholesterol (mmol/L)	4.42±0.65	4.47±0.68	5.15±0.86	5.80±0.73*
LDL (mmol/L)	2.34±0.68	2.52±0.68	3.35±0.86	3.85±0.71
HDL (mmol/L)	1.41±0.18	1.29±0.18*	1.26±0.21	1.12±0.17*
LDL/HDL	1.74±0.51	1.98±0.60	2.75±0.79	3.42±0.74*
apoA1 (g/L)	1.27±0.24	1.11±0.23*	0.95±0.18	0.81±0.13*
apoB (g/L)	0.71±0.17	0.74±0.14	1.03±0.17	1.16±0.20
apoB/apoA1	0.61±0.19	0.67±0.22	1.21±0.40	1.36±0.22*

*P<0.05, P values refer to comparison between non-exposed and exposed cases.

3. Multiple Logistic Regression Analysis

Seven risk factors believed to contribute to CHD were subjected to logistic regression analysis. These factors include: history of hypertension (x1), family history of hypertension (x2), family history of CHD (x3), history of passive smoke exposure (x4), history of drinking (x5), exercise performance test (x6) and history of hypercholesteramia (x7). The results are shown in Table 5.

Table 5. Multiple Regression Analysis of CHD Risk Factors

	Bi	Var(Bi)	S(Bi)	STD(Bi)	OR	G	P
History of Passive smoke	0.406	0.069	0.083	4.87	1.5004	16.93	<0.01
History of Hypertension	0.714	0.052	0.227	3.147	2.0429	8.90	<0.01

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DISCUSSION

To avoid and minimize bias introduced in studies using hospitalized subjects, the present investigations compared the subjects groups to two control groups, one group consisting of patients hospitalized for reasons other than CHDs, and a second group randomly selected from the general population. These two control groups are compared to the CHD-diseased group.

Investigations on the effects of passive smoking in females are more difficult to perform than comparable studies aimed at the effects of active smoking, because the effects of passive smoking may be dependent on such factors as humidity, ventilation and other indoor environmental considerations. Studies to date have not been able to produce a widely accepted standardized protocol for this type of investigation. One of the methods which have been used to assess passive smoke exposure in females relies upon the smoking status of spouses, which have been used in several previous published reports.[1,4,6] The method appears to provide a certain degree of simplicity, feasibility, and relative objectivity.

Female passive smokers have an OR of 3-3.5 in getting CHD, with 95% CI greater than 1. The exposure dose is associated with angina pectoris, in agreement with results of other investigators. The associations remain after adjusting for potential confounders, suggesting that there is a direct correlation between passive smoking and CHD in females. Additionally, our investigations also showed alterations in blood cholesterol and lipoprotein levels, indicating that an alteration in the metabolism of cholesterol and/or lipoprotein could contribute to CHD in female passive smokers. According to Scott et al.[1], 85% of indoor smoke is due to sidestream smoke, which is known to contain a higher concentration of many toxic chemicals than mainstream smoke, and presumably exhibit a more pronounced adverse health effect. Previous studies have shown that an increase in blood COHb levels capable of producing an obviously untoward effect in people with heart and lung diseases.[1] Arrownow studied 10 subjects with angina pectoris, and reported a doubling of blood COHb 2 hours after exposure to indoor tobacco smoke in a poorly ventilated environment. These subjects also showed a 33% reduction in time of exercise before reaching a perceived exertion. The mechanism, however, remains to be investigated.

In People's Republic of China, 33.88% of population age>15 years are smokers and 61% of males regular smokers. The indirect public health consequences of smoking has not received enough attention. Despite the limited number of cases used in the present investigation which obviously have severe restrictions, it suggests that passive smoking is related to CHD in females. Thus, smoking in public should be restricted

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